A Dissertation on

LONG-TERM EFFECTS OF KIDNEY DONATION ON RENAL FUNCTION AND BLOOD PRESSURE

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CERTIFICATE

This is to certify that "**LONG-TERM EFFECTS OF KIDNEY DONATION ON RENAL FUNCTION AND BLOOD PRESSURE**" is bonafide work done by **Dr. JACOB NINAN**, post graduate student, Department of **Internal Medicine**, **Kilpauk Medical College**, Chennai 10 under my guidance and supervision in fulfillment of regulations of The Tamilnadu **Dr. M.G.R. Medical University** for the award of **M.D. Degree Branch I, Part II (General Medicine)** during the academic period from March 2005 to March 2008.

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CONTENTS

Chapter No.	Titles	Page No.
1	Introduction	1
2.	Aim of study	4
3	Review of Literature	5
4	Materials and methods	23
5	Results	29
6	Discussion	55
7	Conclusions	65
8	Appendix Bibliography Questionnaire Master Chart	

AIM

1. To study the effects of renal donation on renal function in the remnant kidney.
2. To study the influence of gender on renal function in kidney donors
3. To evaluate the correlation between body mass index and renal function in kidney donors.
4. To evaluate the correlation between age and renal function in kidney donors.
5. To evaluate the changes in renal size in live kidney donors
6. To evaluate the prevalence of hypertension in live kidney donors

Functional Adaptation to Reduction in Renal Mass

‘The renal tubules behave not as a machine failing and giving out but as a mechanism pushed to the limit of its capacity.’[5]

Robert Platt

Congenital absence of contralateral kidney has demonstrated that animals and man can lead a normal life with only one kidney. Surgical removal of one kidney in experimental animals and patients was performed in the nineteenth century and led to the observation that the remaining kidney subsequently undergoes compensatory growth. However, our understanding of the remarkable adaptive changes in structure and function of the kidney, after reduction in total renal mass, is the result of experimental studies over the past few decades.

After the loss of renal mass, the remaining renal tissue compensates with an increase in growth. The rate of [^{14}C]Choline incorporation into phospholipid of renal cortical slices was increased by 37% 5 min after surgery and persisted for at least 6 days. In addition, both the formation of additional cells (hyperplasia) and cellular enlargement (hypertrophy) are hallmarks of compensatory growth response. Alterations in DNA synthesis preparatory to cell division have been demonstrated within 6 hours after uninephrectomy and cell division reaches a peak at 2 days and again several

days later.[7, 8] An increase in RNA synthesis, used as an index of hypertrophy, is found within the first 12 hours and persists for weeks as renal mass increases. Two weeks after surgery total RNA content rises 40% and total DNA content by approximately 25%, showing that the predominant factor influencing compensatory growth is hypertrophy.[8]

An increase in the weight of the contralateral kidney in the uninephrectomised rat and mouse is demonstratable within 1 – 2 days after surgery, and growth continues until a maximum increase of approximately 40% above control level is achieved within 1 – 2 weeks.[9, 10, 11] On the basis of X-ray studies a marked increase in renal size also occurs in man after nephrectomy.[12] Compensatory renal growth is initiated by loss of viable renal tissue and does not require surgical removal of tissue.

The rate of compensatory growth is stimulated by a number of factors, including a high- protein diet, amino acids, administration of NH_4Cl, and several endogenous hormones.[13] Conversely, starvation, protein depletion, and endocrine abnormalities retard growth. Galla et al. showed that the magnitude of compensatory growth in immature weanling rats was greater than in adults.[14] Other studies have shown that compensatory growth is blunted in old animals, compared with younger adults.[15]

Studies in rats have shown that the extent of compensatory growth correlates closely with the amount of renal tissue that is surgically removed.

Kaufmann et al. compared the rate of growth in residual kidney tissue over 4-wk interval after loss of 50% and 70% of renal mass with pair-fed controls.[16] The weight of the remaining renal tissue increased by 81% in uninephrectomised animals and 168% in rats with surgical ablation of 70% of the initial mass, compared with an increase of 31% in controls. The formation of new cells, by hyperplasia corresponds in general with the increase in renal mass. After uninephrectomy and two- thirds nephrectomy there is an increase of 25% and 87% in new cell formation in the cortex of the contralateral and remnant kidney, respectively.[17]

Hayslett et al. determined tubular length, luminal diameter, and cell volume in proximal and distal convolutions from normal and unilaterally nephrectomised rats. There was an increase of approximately 15% in the luminal and external diameters of proximal tubule and a 35% increase in length. The distal tubule increased about 10% in diameter and 17% in length. Both luminal and cell volumes of proximal tubule approximately doubled, whereas in the distal convolution the increase was in order of 50% for luminal volume and 25% for cell volume.[18] Oliver et al. demonstrated predominance of compensatory growth in the proximal tubule convolution, including a marked increase in length of pars recta and loop of Henle, in three-quarters nephrectomised rats.[19] There is experimental evidence that compensatory growth occurs relatively symmetrically throughout the cortex.

In both uninephrectomised animals and after 70% nephrectomy the hypertrophy index was equal in outer, middle, and deep zones of the cortex.[20]

Two hypotheses had been invoked to explain the phenomena of compensatory renal growth. The first attributes compensatory growth changes in the remaining kidney to the increased work load that the smaller renal mass is called on to perform. Studies have shown conclusively that this mechanism does not apply. Diversion of the urinary stream from one kidney into the peritoneal cavity results in a 30% increase in glomerular filtration rate and concomitantly in increased net reabsorption of sodium by the contralateral kidney. There was no evidence for an increase in renal growth 4 days after surgery, a time interval sufficiently long to demonstrate compensatory growth if it were to occur.[21] According to the second hypothesis, compensatory growth is attributed to an organ-specific humoral substance that may control renal mass. The role of a humoral mediator is supported by evidence that compensatory renal growth occurs in heterotopic kidney transplanted to an anephric host and in normal animals connected by parabiosis with an anephric partner.

As the population of nephrons diminishes, while the dietary intake and/or endogenous production of water and solutes is unchanged, there is a proportional increase in the excretion of water and solute by individual

residual nephrons. This adaptive change, which preserves zero net balance in the early phase of renal insufficiency, involves a reduction in the fractional reabsorption of substances derived from the initial glomerular ultrafiltrate and an increase in the rate of secretion of solutes that are extracted by tubular epithelial cells from peritubular blood. These compensatory changes are adequate to maintain electrolyte and water homeostasis until severe renal failure ensures (GFR < 20% of normal).[22]

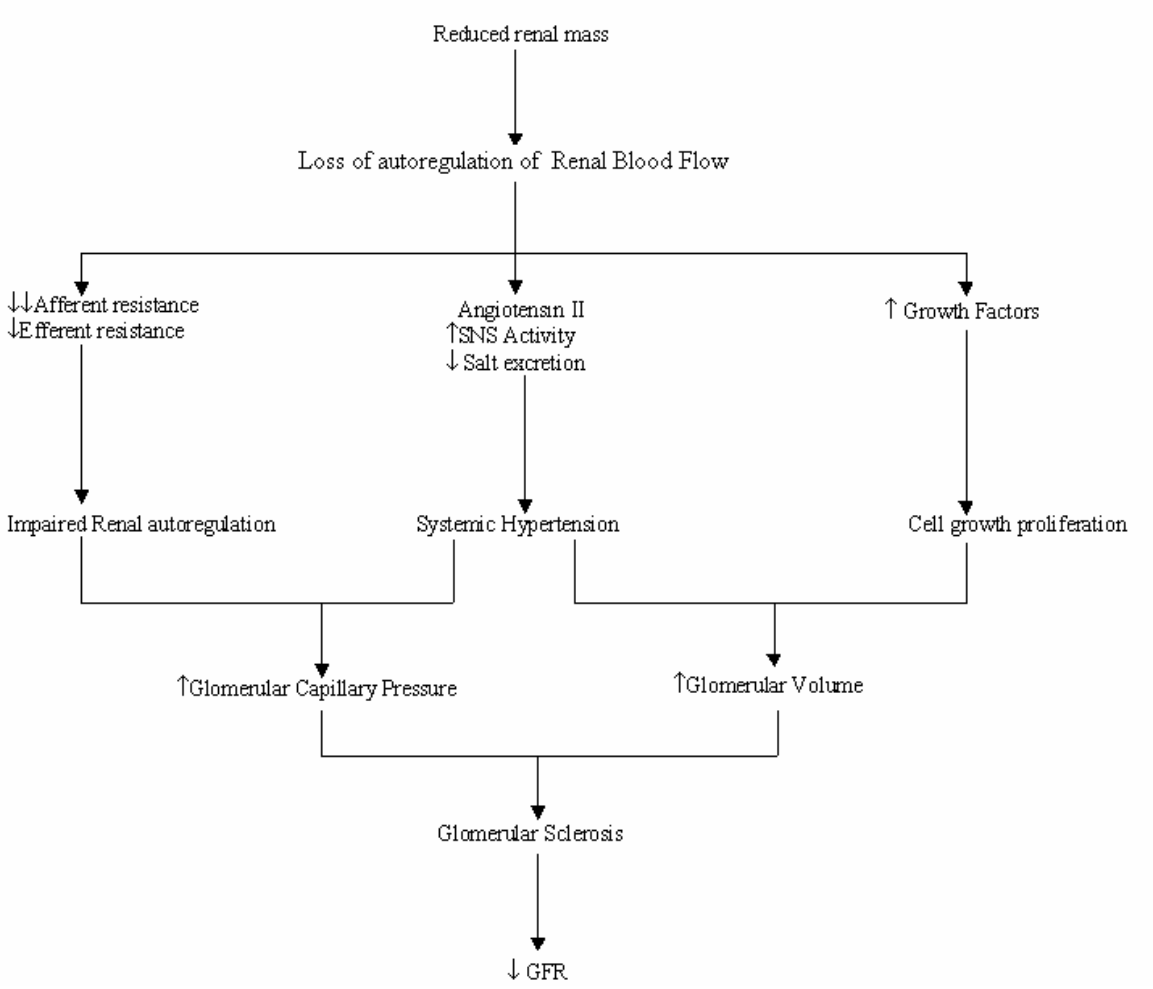
The Hyperfiltration theory

Experimental studies incriminate glomerular hypertension in mediating progressive renal damage after any of a variety of initiating injuries. Glomerular hypertension and hyperfiltration occur in humans with diabetes mellitus, solitary or remnant kidneys, and various forms of acquired renal disease. Without hyperfiltration, serum creatinine levels would double as clearance decreases by 50% when half the total renal mass is removed.

Brenner and colleagues proposed that maladaptive glomerular changes exert a major influence on the influence on the factors that initiate and perpetuate disease progression. These hemodynamic changes lead to glomerular hyperfiltration, an adaptation seen in response to a reduction in functional nephron number whether induced genetically, surgically, or by

acquired renal disease. The elevated single nephron glomerular filtration rate (SNGFR) common to these pathophysiologic conditions is usually caused by increases in glomerular capillary plasma flow rate (Q_A) and mean glomerular capillary hydraulic pressure (P_{GC}), which in turn are due to adaptive reductions in preglomerular and postglomerular arteriolar resistances. Progressive glomerular sclerosis and proteinuria eventually occur in most experimental models of renal disease characterized by glomerular hyperfiltration and hypertension

Altered Glomerular hemodynamics in humans



Azar et al. and Olson et al. demonstrated that in experimental hypertension, unassociated with primary renal disease, impaired autoregulation of renal blood flow (RBF) and glomerular flow rate (GFR) is decreased afferent arteriolar resistance (RA) causes hydraulically mediated renal damage and progressive glomerular injury.[23, 24, 25]

Experimental studies in dogs and rats with a marked reduction in renal mass exhibit significant loss of autoregulation of RBF and GFR in response to changes in renal perfusion pressure.[26]

In rats with subtotal renal ablation or in uninephrectomized spontaneously hypertensive rats, increases in systemic pressure are transmitted directly to glomerular capillaries as the result of a greater reduction in RA compared with efferent arteriolar resistance (RE).[27, 28, 29] The consequent increment in P_{GC} is associated with progressive proteinuria and glomerular injury.

The relation between intraglomerular hemodynamic abnormalities and progressive glomerulosclerosis in experimental renal disease is not straightforward. Yoshida et al. [30, 31] demonstrated that glomerulosclerosis correlated with glomerular hypertrophy and not PGC in a rat remnant kidney model. Similarly, other investigators have dissociated progression of experimental renal disease from increments in PGC and have suggested that measures of nephronal hypertrophy such as VG and RGC or the production of growth factors are more tightly linked to ongoing glomerular injury.[32, 33, 34, 35, 36] In this regard, mesangial cells in culture proliferate[37] and produce excess type I and IV collagen[38] in response to mechanical stretching. Further, both angiotensin II[39, 40] and endothelin[41, 42] stimulate mitogenesis of cultured mesangial cells. The degree of glomerular sclerosis correlates significantly with urinary excretion of endothelin in rats with subtotal renal

ablation.[42] Consequently, the above data imply that in various experimental models renal disease progresses by different mechanisms; increments in PGC or VG or augmented production of angiotensin II or endothelin stimulate the production of growth factors that results in progressive glomerular obsolescence.

The influence of hyperfiltration, the most readily monitored surrogate of altered glomerular hemodynamics, on renal function in humans has been most thoroughly evaluated in kidney transplant donors and in patients uninephrectomised for acquired renal disease. Even after one to two decades, total GFR often averages approximately 70% of prenephrectomy values despite the 50% reduction in renal masses, indicating that the remaining kidney is indeed hyperfiltering. Most studies have shown that in association with this hyperfilteration the prevalence of hypertension in both groups tends to increase after uninephrectomy, although only in transplant donors is hypertension apt to be more prevalent than in local, control populations. Similarly, the prevalence of proteinuria tends to increase in groups after uninephrectomy, proteinuria was shown to increase as a function of time after surgery.

Long-term effects of reduced renal mass in humans

In early stages of permanent renal injury or extensive ablation, structural and functional adaptations associated with hypertrophy partially compensate for nephron losses. Glomerulotubular balance is maintained in these conditioned nephrons by intrinsic tubule and peritubular capillary adaptations that parallel single nephron glomerular filtration rate (SNGFR). Studies of Na^+-H^+ exchange in renal cortical brush border membrane vesicles indicate that tubule functional adaptation is not tied to loss of renal mass per se but rather to factors such as dietary protein content that set the level of SNGFR. Likewise, the structural heterogeneity that follows chronic renal injury or extreme ablation of renal mass is less a consequence of nephron injury than of adaptation linked to dietary protein intake.

As the population of nephrons diminishes, while the dietary intake and/or endogenous production of water and solutes is unchanged, there is a proportional increase in the excretion of water and solute by individual residual nephrons. This adaptive change, which preserves zero net balance in the early phase of renal insufficiency, involves a reduction in the fractional reabsorption of substances derived from the initial glomerular ultrafiltrate and an increase in the rate of secretion of solutes that are extracted by tubular epithelial cells from peritubular blood. These compensatory changes are

adequate to maintain electrolyte and water homeostasis until severe renal failure ensures (GFR less than 20% of normal). After a moderate reduction in nephron population there is no evidence that the factors that modulate ion transport are qualitatively different from those that regulate renal function in the intact subject, when the excretory load of solute is varied by changes in intake or endogenous production.

Long-term risks after living kidney donation

Over the past four decades, the surgical technique of harvesting the donor kidney has been improved to such an extend that the perioperative mortality has been reduced to 1 out of 3000 living donor nephrectomies.[43] In the 1980s with reports of progressive hyperfiltration-mediated structural damage to remaining nephrons in experimental animals, there has been great concern and debate on the long term prognosis of kidney donors.

Although increased long-term mortality has not been reported in healthy persons after nephrectomy.[44, 45, 46] A survival benefit was demonstrated among donors in a large Norwegian series, which has been attributed to the rigorous evaluation and screening methods prior to renal donation. In a metaanalysis that took 48 studies and 5048 donors into evaluation, only 1 study showed 2 donor deaths due to renal failure. Seven

studies described cardiovascular disease in a proportion of living renal donors.[47]

Long term effects on renal function, blood pressure, proteinuria/albuminuria and occurrence of ESRD has been studied in great detail worldwide.

Renal function

Accurate assessment of renal function is an essential component of donor evaluation. 24-hr urine collection for creatinine clearance (CCr), iothalamate clearance, chromium 51 – labeled ethylene diaminetetra-acetate (Cr^{51} – EDTA), measurement of cystatin C or calculated estimate using Cockcroft-Gault formula or the Levey formula has been used for the estimation of GFR. After donor nephrectomy, serum creatinine levels increased by approximately 25% and creatinine clearance falls by approximately the same percentage.[47] In a metaanalysis by Garg AX et al. an average decrement in GFR by 26ml/min (per $1.73m^2$) with the average GFR being 86ml/min (per $1.73m^2$).[48] In an Indian pilot study by Sahay M et al. a reduction of 28.2 +/- 13.57 ml/min, a decline of GFR by 17 ml/min immediately post nephrectomy.[49] This was followed by stabilization of GFR with subsequent rise by 1.4ml/ min/ decade. In reports from Sweden and the

United States no statistical evidence of rapid decline in GFR than the expected population was demonstrated.[48] Several studies indicate that functional adaptation occurs rapidly after uninephrectomy with GFR remaining stable over many years. The Swiss Organ Living Donor Health Registry (SOL-DHR) data demonstrated a slow improvement for measures of serum creatinine and creatinine clearance.[47] This finding is in contrast with the expected physiological decline in GFR associated with ageing (i.e., approximately 1mL/min/year). Thus, the effect of nephrectomy in terms of GFR by hyperfiltration outweighs the effect of normal ageing, atleast during the first decade. The trend beyond the first decade after nephrectomy and whether it may over time result in adverse changes within the remaining kidney has yet to be answered by long term prospective studies.

Proteinuria

Most progressive renal disease and all advanced glomerular diseases are accompanied by proteinuria and albuminuria. Albumin has been suggested as the better variable for follow up in donor nephrectomies as it is a single, relatively small protein, which can be measured fairly accurately and the earliest to appear in urine in the setting of renal damage. Slight increase of glomerular intracapillary pressure or minimal ‘glomerular

hypertension' is expected in post nephrectomy patients. As glomerular injury progresses, systemic and glomerular hypertension may worsen and accelerate renal damage. Since microalbuminuria is a sign of glomerular injury, its recognition and treatment are important if progressive damage is to be avoided.

Garg AX et al. in his metaanalysis showed an incidence of proteinuria in 42 studies to be ranging from less than 5% to 20%.[48] The pooled incidence of proteinuria among 9 studies which quantified the proteinuria in a total of 1799 donors for 7 years was 10%. There was a statistically significant increase in 24-hr urine protein in donors when compared to controls an average of 11 years postnephrectomy.[49] In two of the studies, 24-hr urine albumin was found to be 56 mg higher in donors compared to controls 14 years after donation. The pooled risk of microalbuminuria after kidney donation was 3.9.

Statistically significant development of proteinuria in post nephrectomy patients where reported by Sahay et al. 40% of the population in study developed microalbuminuria and 14% developed overt proteinuria.[49] In a study by Fehrman-Ekholm et al. demonstrated that donors with proteinuria were more prone to hypertension and had lower GFR than donors without proteinuria. Data from a number of studies appear to indicate

a significant increase in development of proteinuria in donors when compared to the general population.

In Switzerland, donors who develop albuminuria are advised to consult their family physicians for the initiation of renoprotective drugs such as ACEIs and ARBs.

Hypertension

Hypertension remains an issue of concern in kidney donors as it may initiate or accelerate nephrosclerosis and renal failure in the normal solitary kidney. Renal reserve is reduced even if serum creatinine is within normal levels.[50] Most investigators have reported hypertension in 17 – 33% of former donors. When these results were compared with age-matched general population there was no significant incidence of hypertension.[51, 52, 53, 54] In a metaanalysis of all studies comprising more than 3100 nephrectomised patients and 1700 appropriate controls, no increment in incidence of hypertension was found. However, there was an average increase in blood pressure of 2 – 3 mmHg and a further increase in systolic pressure of 1mm Hg for each decade following kidney ablation. In Sahay M et al. study a statistically significant 9.96 mm Hg rise in mean arterial pressure was

reported.[49] All donors with a family history of hypertension became hypertensive post nephrectomy.

Occurrence of End Stage Renal Disease in Donors

Functional and anatomical adaptation to reduction renal mass is achieved by the remaining nephrons, unlike living liver donation where liver regeneration occurs up to 80%. Studies in experimental model have demonstrated hyperfiltration, albuminuria and ultimately renal insufficiency following ablation of renal mass. It is possible that similar events may occur in human kidney donors.

Adaptive hyperfiltration has found to stabilize the clearance at 70 – 80% of pre-donation values in many studies. Albuminuria the earliest indicator of glomerular hypertension reflects the changes in selectivity of glomerular permeability, ultimately resulting in injury. Continuous renal insult leads to slow attrition of nephron numbers, commonly termed ‘hyperfiltration injury’ leading to focal and then global glomerulosclerosis in the remnant kidney.

In 2002, United Network for Organ Sharing (UNOS) database analysis of 47996 living donors showed that 20 donors had been listed for cadaveric kidney transplantation. Another 36 donors had been transplanted

before UNOS was started in 1987. Interestingly 85% of the subjects had donated to a sibling, indicating the possibility of a genetic predisposition to kidney disease. The time from donation to ESRD in these subjects ranged from 2 to 31 years.[55] Hypertensive nephrosclerosis, focal glomerulosclerosis and chronic glomerulonephritis accounted for two-thirds of diagnoses leading to ESRD. In Garg AX et al metaanalysis, a total of 10 donors from eight different studies were living with kidney failure at the time of late assessment.[48] In a case series from Chennai by Prakash KC, a total of ten renal donors who developed renal complications were seen. Three donors had acute renal failure with complete recovery of renal functions. Two donors had nephrotic syndrome of which one responded completely to a short course of steroids. Five donors developed chronic renal failure (One was on dialysis support, one underwent renal transplantation and three are on conservative management).[56]

Current data are inconclusive but might be interpreted to indicate that the occurrence of ESRD is higher after living donor nephrectomy than in the general population. However, since most of the donors are relatives of someone with kidney diseases, the actual increment in risk may be difficult to calculate.

Additional studies and lengthier follow-up using the existing databases will be necessary to be able to provide accurate information about the risk of developing ESRD to prospective donors.

The currently available data indicate that long-term health risks associated with donor nephrectomy are quite low. Donors should be rigorously evaluated before the nephrectomy and followed up frequently to detect changes in blood pressure, albumin excretion and renal function. Early detection and appropriate medical or surgical intervention is of utmost importance as it generally gives the best chance for preventing deterioration in health or ESRD.

MATERIALS AND METHODS

SETTING

A retrospective cohort study and analysis of patients drawn from Nephrology Clinic in Government Kilpauk Medical College,Kilpauk and Government General Hospital, Chennai.

STUDY POPULATION

The study was conducted over a 12 month period from June 2006 to June 2007. Cases were drawn from Nephrology clinic, Department of Nephrology, Kilpauk Medical College and Govt. General Hospital, Chennai. Informed consent was taken from all participants of the study. A total of fifty-three patients were randomly chosen at the start of the study. Ten patients were excluded from study, as they were not willing for any investigations. Five patients were omitted as they were not able to provide pretransplantation records and details. Five patients did not report after basic renal blood and urine investigations.

INCLUSION CRITERIA

All live kidney donors

EXCLUSION CRITERIA

- Patients unwilling for investigations
- Patients allergic to nuclear tracer drugs
- Patients with recent history suggestive of ARF
- Patients with CCF
- Patients with known congenital renal diseases (viz., Polycystic kidney disease, Alport's disease, etc)
- Patients on long term use of analgesics
- Patients who are known diabetics/HbsAg/HIV

Information was collected as per the Performa (Appendix I), consisted of basic data, including name, sex, age and occupation. All patients were from low socio-economic groups according to Kuppuswamy Scale.[57] Detailed history pertaining to symptoms suggestive of renal failure, ischemic heart disease and renal stone disease was recorded from the patients. Patients were enquired regarding the usage of antihypertensive drugs or any other medications.

Anthropometric measurements including height in metres (standing height using stadiometer) and weight in kilograms were measured.

Vitals were recorded. Blood pressure was recorded at three occasions during the course of the study.

Detailed clinical examination of all systems was carried out to rule out any associated disease state.

Predonation random blood sugar, blood urea, serum creatinine, serum electrolytes and ultrasound measurements were recorded from the donors previous records.

The donors underwent serum random blood sugar, blood urea, serum creatinine and serum electrolytes measurement. The post donation glomerular filtration rate was calculated using these data according to the Cockcroft Gault and MDRD (IV) formulae. The measurement of the undonated kidney was performed using ultrasonography. The donors were then taken up for ^{99m}Tc DTPA Renal Scintigraphy for estimation of glomerular filtration rate.

REFERENCE VALUES USED IN THIS STUDY

BMI (WHO criteria for Asian population)[58]

Body Mass Index = Weight (kg) / Height $(m)^2$

Values 18.5 -22.9 kg/m^2 was taken as normal weight

23 – 29.9 kg/m^2 was taken as overweight

≥ 30 kg/m^2 was taken as obesity

BODY SURFACE AREA (BSA)

The Mosteller formula was used for calculation of body surface area[59]

BSA (m^2) = ([Height(cm) x Weight(kg)]/ 3600)^{½}

STAGING OF HYPERTENSION (According to JNC VII)[(102)]

Classification	Systolic BP (in mmHg)	Diastolic BP (in mmHg)
Normal	< 120	< 80
Pre-hypertension	120 – 139	80 – 89
Stage I	140 – 159	90 – 99
Stage II	≥ 160	≥ 100

BLOOD SUGAR

Fasting Glucose ≥ 126 mg % and random blood sugar ≥ 200 mg% patients with symptoms were taken as diabetes. Such patients were excluded from the study..

BLOOD UREA

The Blood urea in this study was estimated using **DAM** method (Diacetyl Monoxime). The normal value which being 20 – 40 mg%.

SERUM CREATININE

Serum creatinine was estimated using Modified Jaffe's method. Values of >1.5mg%[60] in males and females were suggestive of renal impairment.

SERUM ELECTROLYTES

Serum sodium and potassium were measured using flame spectrometric method. Serum sodium values of 136 – 145 meq/L were considered to be within the normal range. Serum potassium values of 3.5 – 5 meq/L were considered within normal range.

ULTRASONOGRAPHY

The kidneys were visualized using a Larsen Tourbo Ltd. Sonata Plus Ultrasonogram in GKMCH Department of Radiology and measurements taken as accurately as possible.

^{99m}Tc DTPA RENAL SCINTIGRAPHY

Renal Scintiscan was performed after i.v injection of 5.0 mCi ^{99m}Tc – DTPA. Early dynamic images were acquired for 1 minute and delayed static images were acquired after 20 minutes. 20mg Furosemide i.v was administered along with the activity – (F + 0) protocol. The images were then processed using the GATES algorithm.

STAGES OF CHRONIC KIDNEY DISEASE [61]

Stage	Description	GFR (ml/min/1.73m^2)
1	Kidney damage with normal or ↑ GFR	>90
2	Kidney damage with mild ↓ GFR	60-89
3	Moderate ↓ GFR	30-59
4	Severe ↓ GFR	15-29

DISCUSSION

In this study, done among 30 renal donors the mean age of the renal donors at evaluation was 41.7 yrs ± 7.535 (Mean ± S.D). In comparison to many of the other studies this study has a younger population.

Study	Mean	Range
This study	41.7 yrs ± 7.535	27 – 58
Saran R et al. [62]	64 yrs ± 9	38 – 80
Talseth T et al. [63]	56 yrs	42 – 66
Grossmann J et al. [64]	57 yrs ± 11	NA

Although older age at the time of donation was associated with lower pre- and post donation GFR, no statistical association between change in GFR after donation and age at the time of donation was reported by AX Garg et al..[48] A significant association was demonstrated between fall in GFR and age of donors at nephrectomy in this study. The mean age of the donors at donation in this study was 37.1 yrs ± 8.932 (Mean ± S.D). This study constituted of a population younger than other studies under comparison.

Study	Mean	Range
This study	37.1 yrs ± 8.932	20 – 55
Talseth T et al. [63]	46 yrs	33 – 55
Grossmann J et al. [64]	45 yrs ± 11	NA
Sahay M et al.[49]	41 yrs ± 8.12	25 – 54.16

The gender distribution demonstrated a majority of 80% female donors compared to a 20% donation by the opposite sex.

Study	Males	Females
This study	20	80
Saran R et al.[57]	49	51
Grossmann J et al.[59]	29	71
Sahay M et al.[49]	56	44

In comparison to western studies, in which the mean duration following donation was greater than a decade, this study had shorter duration of follow-up. The mean duration since nephrectomy was 4.69 yrs +/- 4.288

(Mean ± S.D). The mean follow-up duration of this study was similar to the pilot study conducted in Hyderabad.[49]

Study	Mean	Range
This study	4.69 yrs ± 4.288	0.6 – 15
Saran R et al.	19.6 yrs ± 5.0	NA
Talseth T et al.	11	9.9 – 12.25
Grossmann J et al.	11 yrs ± 7	1 – 28
Sahay M et al.	5.25	NA

Praga M et al. demonstrated renal insufficiency after renal donation in 92% of donors with BMI >30kg/m^2 at the time of nephrectomy. Multiple logistic regression analysis showed the risk of developing renal disease statistically correlated with BMI at the time of nephrectomy.[65] In comparison to Grossmann et al., whose mean BMI was 26 ± 4; this study had a lower BMI of 24.84 kg/m^2 ± 4.024.

Goldfarb et al. reported a higher serum creatinine after donation for males but no difference in GFR.[66] A similar result was obtained in this study. A significantly higher serum creatinine was observed in males

compared to females while no difference was noted between the genders and post donation GFR.

The minimal data regarding human glomerular hemodynamics suggest that reduced pre-glomerular vascular resistance to be responsible for the hyperfiltration that occurs after nephrectomy.[67, 68] Without hyperfiltration, serum creatinine would double as GFR decreases by 50% when half the total renal mass is removed. After donor nephrectomy, serum creatinine levels increase by approximately 25% and GFR falls by approximately the same percentage.[47] The Swiss Organ Living Donor Health Registry (SOL-DHR) data indicate a slow improvement for measures of serum creatinine and GFR. In a meta-analysis by AX Garg et al., the average serum creatinine before and at follow-up was 0.92mg/d and 1.11mg/dl respectively.[48] In this study the rise in serum creatinine was observed to be 16% and the reduction in GFR (estimated by MDRD formula) to be approximately 17%. The result that there is a statistically significant increase in serum creatinine following kidney donation was corroborates with the findings of Grossmann et al. [64]

Study	Predonation S. Creatinine	Post donation	Significance
This study	0.915 mg/dl ± 0.073	1.069 ± 0.284	P = 0.0065
Sahay M et al.	0.97 ± 0.09 mg/dl	1.22 ± 0.82	Ns
Talseth T et al.	79 μmol/l	88	NA
Grossman J et al.	72.5 ± 15 μmol/l	85.7 ± 16.8	P < 0.001

Sahay M et al., studied donor sex influence on renal function and demonstrated a significantly greater degree of hypertrophy in males as compared to females. Although females were observed to have greater degree of hypertrophy in comparison to males (7% vs 5%), the increase was not found to be statistically significant according to this study. There was a statistically significant increase in the renal length and width of the remnant kidney, which corroborated with Sahay M et al. study.[49]

Study	Predonation Kidney length	Post donation	Significance
This study	9.804	10.536	P < 0.001
Sahay M et al.	9.46 ± 0.39	10.60 ± 0.73	P < 0.05

A meta-analysis by AX Garg et al., the mean GFR before nephrectomy and at follow up was 111ml/min and 86 ml/min, respectively.[48]

A meta-analysis by Kasike et al. showed a decline of GFR by 17ml/min immediately post nephrectomy.[69] This was followed by stabilization of GFR with subsequent rise by 1.4ml/min/decade. The Swiss Organ Living Donor Health Registry (SOL-DHR) data also demonstrated a slow improvement for measures of serum creatinine and GFR. This finding is in contrast with the expected physiological decline in GFR associated with ageing (i.e., approximately 1ml/min/year). Thus, the effect of nephrectomy in terms of increasing GFR by hyperfiltration outweighs the effect of normal renal ageing, at least during the first decade. The as-yet-unanswered questions are whether this trend will continue beyond the first decade after nephrectomy and whether it may, over time, result in adverse changes within the remaining kidney. There is however data from Sweden and Cleveland that demonstrated no significant fall in GFR and a stabilization of GFR to approximately 72% of predonation levels at least 25 years after donation.[66, 70] The solution to such unanswered questions lies in prospective data collection or registries and long-term studies (including patients beyond 40 years postnephrectomy).

In studies by Saran R et al. and Goldfarb DA et al., although women were reported to have lower GFR both before and after donation when compared to men, no statistically significant gender difference in decrement

in GFR after donation was observed in this study.[62, 66] Elderly donors demonstrated a lower GFR both before and after donation; the decrement in GFR after donation, however, tended to be smaller, larger or no different than younger individuals in many studies. Although Gracida et al. reported predonation obesity not to be associated with post-donation GFR; Praga et al reported statistical correlation between developing renal disease and BMI at the time of nephrectomy. [65, 71] The time after donation was not associated with post-donation GFR or change in GFR in this study. In an isolated study, a higher drop in GFR was associated with higher pre-donation blood pressure [63] and higher predonation GFR.[64] The latter observation was explained by Talseth T et al as a regression towards the mean rather than a true reduction in renal function.

A statistical significant reduction in GFR (calculated by MDRD) after nephrectomy was demonstrated in this study. In this study statistically significant association was established between fall in GFR and age at donation, gender, body mass index, body surface area and predonation GFR.

Study	Predonation GFR (MDRD)	Postdonation GFR	Significance
This study	80.367 ± 15.343	66.533 ± 17.797	0.0012
Grossmann et al.	92 ± 20	71 ± 15	P <0.001

The development of hypertension following kidney donation was found to statistically significant in this study like in other studies like Grossmann et al. and Sahay M et al.[49,64]

Study	Predonation incidence of HT	Post donation	Significance
This study	0%	33%	P < 0.001
Talseth T et al.	10.29%	14%	Ns
Grossman J et al.	7%	30%	P < 0.001
Goldfarb et al.	0%	48%	P < 0.001
Sahay M et al.	0.5%	46%	P <0.05

Most authors have reported an increased prevalence of hypertension (4% to 31%) in kidney donors. Although many studies found statistically significant increase in prevalence of hypertension following donation, this association was found not statistically significant when compared to an age

matched population. This could be explained by the tendency of the human hemodynamics to develop hypertension with aging. Further more, the increase in systolic blood pressure seen in donors older than 50 years likely reflects the age related increase in blood pressure noted in the general population. [66]

Grossmann et al. reported a negative correlation between blood pressures at the time of donation and increase in blood pressure over time, i.e., the lower BP at the time of donation, the larger the increase over time. [64] Talseth et al., reported a positive correlation between pre-operative and follow-up blood pressure and suggested that the relatively depressed GFR contributed to the increase in BP postoperatively.[63] Similarly, Eberhard et al. in a study of 29 donors found hypertension in 29% at 11.3 +/- 8 years of follow up.[72] In contrast to the above studies, Mayo Clinic reported no adverse effects on blood pressure in 24 mildly hypertensive donors 1 year after donation.[73] The prevalence of hypertension was not found to be higher than the general population by Grossmann et al..[64] Sahay M et al. demonstrated 46% of the study population to be hypertensive after nephrectomy.[49] Although Talseth et al. reported statistically significant increase in both systolic and diastolic blood pressure, Sahay M et al found this observation to be statistically insignificant in their study.[49, 63] Torres et

al. opinioned that the increased risk for hypertension was due to familial genetic factors predisposing them to hypertension or renal disease, which was proven by the demonstration of hypertension in all donors with a family history by Sahay M et al..[52]

In this study a 33% prevalence of hypertension was noted following donation, which was found to be statistically significant. No significant association was noted between hypertension and gender, postdonation period, reduction in GFR and body mass index. Body surface area and age at donation was found have significant association with the development of hypertension following donation.

The exact number of donors who develop ESRD is unknown, but the incidence appears to be quite low. Several studies show incidence of ESRD to range from 0.04% to 0.64%.[55, 70, 73, 74] Current data are inconclusive but might be interpreted to indicate that the occurrence of ESRD is higher after living donor nephrectomy than in the general population. In this study only one patient was seen to be in stage 4 chronic kidney disease.

CONCLUSION

In this study, the significant changes in renal size, serum creatinine, glomerular filtration rate and prevalence of hypertension shows that Indian kidney donors do not behave any differently from their Western counterparts. Untreated hypertension is a known risk factor for nephrosclerosis and renal failure in the general population. It seems logical that the solitary kidney has enhanced risk and the glomeruli are exposed to greater systolic blood pressures than those of hypertensives with two kidneys. Since hypertension is one of the root causes for many adverse consequences affecting many organs including the kidney, aggressive screening and treatment is indicated. A complete disclosure of all possible short-term and long-term complications after renal donation should be made to every potential donor.

Although currently available data from numerous studies indicate that long-term health risks associated with donor nephrectomy are quite low, arguments for and against organ donation by living have remained for many years because of insufficient thoroughly evaluated data through lack of prospective studies. This is at least partially a direct consequence of using

only healthy persons as donors, and it seems important going forward to preserve this precedent.

This study has shown a significant association between age and serum creatinine at the time of donation and the reduction in glomerular filtration rate. Although no criteria can accurately predict pre-operatively which donor will need medical care and treatment after nephrectomy, the use of fixed cut-off points for donor acceptance, such as age, glomerular filtration rate and blood pressure can help in selection till concrete evidence-based protocols, drawn from long-term prospective studies, for donor selection is made.

Since a renal donor logically conforms to the definition of chronic kidney disease a stage-wise approach for the treatment of donors is essential. The key to a healthy donor in the long run at present lies with strict rigorous screening of donors and strict adherence to “The Consensus Statement of the Amsterdam Forum on the Care of the Live Kidney Donor”.[75]

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Name Age
Sex Occupation
Income No. of family members(family size)
Per capita income

History
Year of donation of kidney
Donated to
C/o pedal edema
C/o facial puffiness
C/o dec. urinary output
C/o burning micturition
C/o loin pain/stones in urine
C/o chest pain/palpitation/dyspnoea/syncope
C/o fever in recent past
H/o diabetes/hypertension/prolonged drug use

O/E
Built – Poor / Moderately / Well
Nourished – Emaciated / Poor / Moderately / Well / Obese
Wt. Ht.
Pallor / Icterus / Clubbing / Cyanosis / Lymphadenopathy / Pedal edema
Pulse BP
Febrile

RS – NAD / Creps

CVS – NAD / Murmurs

P/A – Abdomen – Distended / Scaphoid
Free fluid
Spleenomegaly / Hepatomegaly
Renal Bruit

CNS – NFND

Inv.

	Pre transplantation	Post transplantation
RBS		
B.Urea		
S.Creatinine		
S. Electrolytes Na/K		
Urine routine		
USG Abdomen		
DTPA GFR		

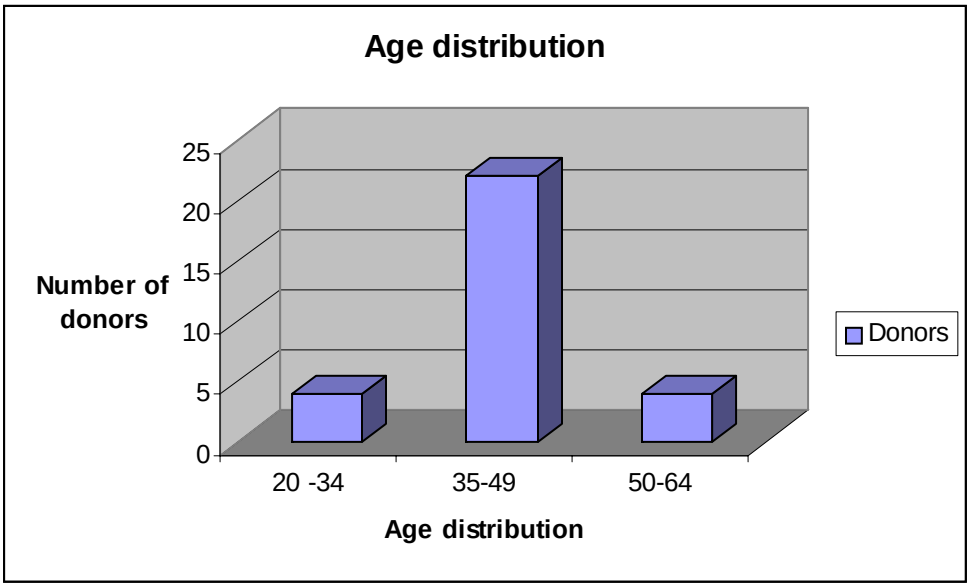
RESULTS

- The Study included 6 males and 24 females.
- In this study the age of the renal donors ranged from 27 to 58 yrs and the age of the renal donors at the time of donation ranged from 20 to 55 yrs.
- The most common recipient of the kidney was the son.
- The study included a majority of 26 patients in their first decade after donation and 4 patients in their second decade after donation.
- 4 patients in the study were found to be obese.
- The number of donors on antihypertensive drugs was 4 and the number of newly detected hypertensives was 6.Therefore, 10 patients out of 30 patients developed hypertension following renal donation.
- 6 patients had predonation GFR greater than 90ml/min/1.73m^2 body surface area while the majority had their predonation GFR between 60 and 89ml/min/1.73m^2.
- 24 patients developed a reduction in GFR as calculated by the MDRD formula, 4 had an increase in GFR and 2 had no change in GFR.
- 1 patient was under conservative management for renal failure following donation after 9 years.

- 28 donors underwent the Technetium^{99} DTPA scan for GFR estimation, 2 donors were unwilling to have the investigation performed.

Age-wise distribution of donors

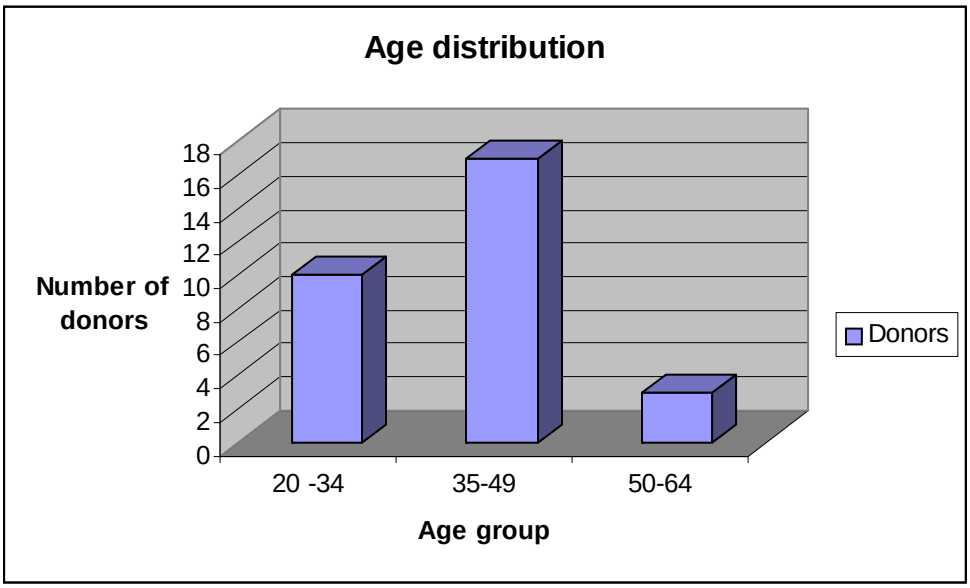
Age Group	Donors
20 - 34	4
35 - 49	22
50 - 64	4



The mean age of renal donors was 41.7yrs ± 2.69 (Mean ± Confidence Interval).

Age-wise distribution of donors at the time of donation

Age Group	Donors
20 - 34	10
35 - 49	17
50 - 64	3

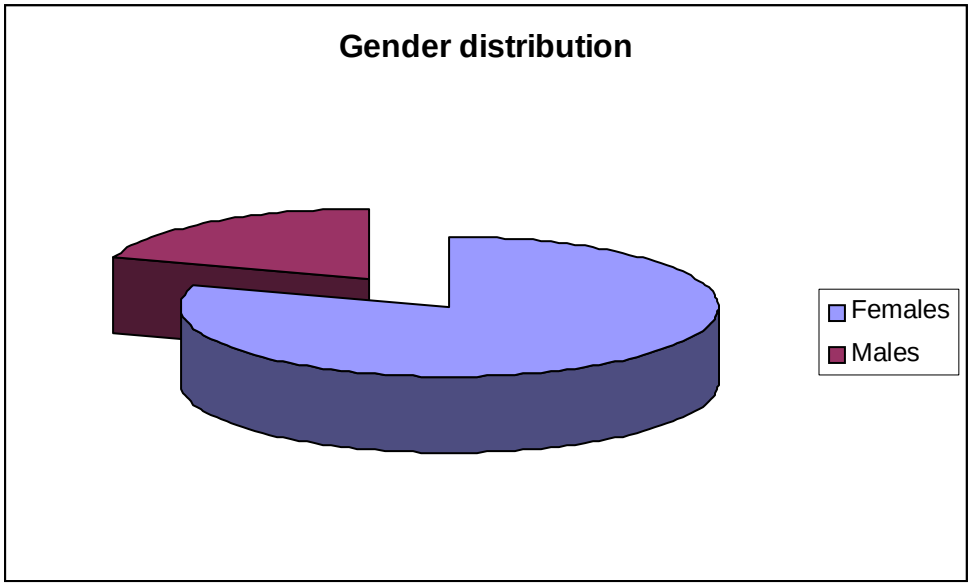


The mean of age at which donors donated their kidney was 37.1yrs ± 3.20

Sex	Mean age	Mean age at donation
Male	41.7 ± 6.029	37.067 ± 7.147
Female	41.58 ± 2.729	37.417 ± 3.218

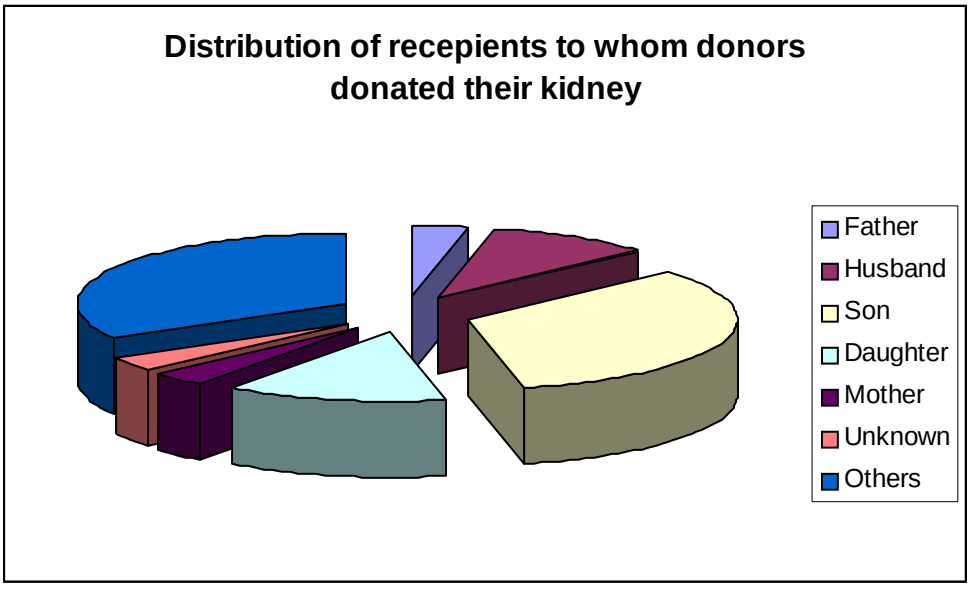
Gender Distibution

Gender	Donors
Females	24
Males	6



Distribution of recipients

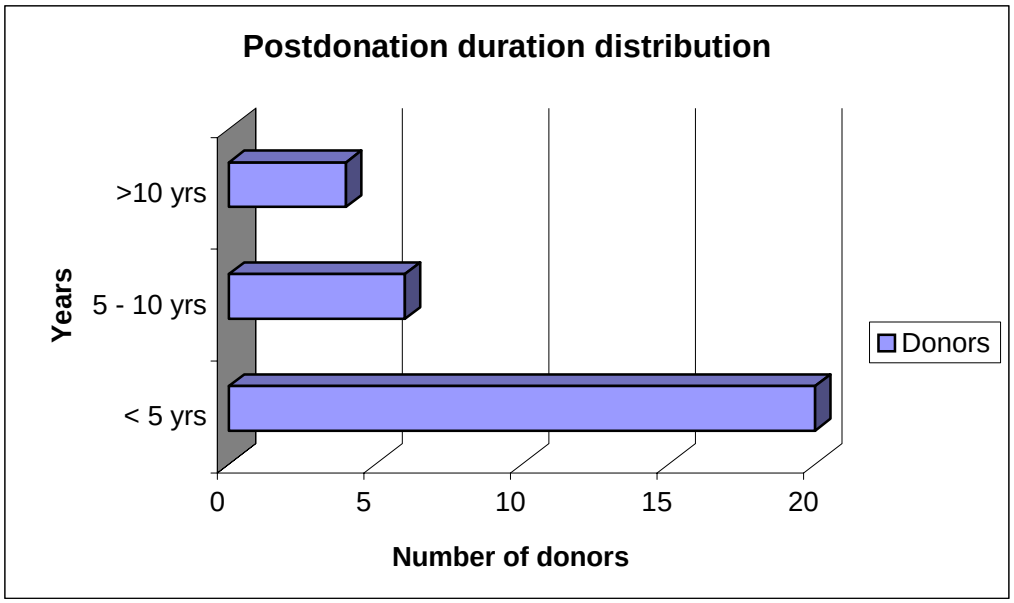
Recipients	Number
Father	1
Husband	3
Son	10
Daughter	4
Mother	1
Unknown	1
Others	10



Distribution of Postdonation duration

Post donation duration	Donors
< 5 yrs	20
5 - 10 yrs	6
>10 yrs	4

The post donation period ranging from .6 – 15yrs.The mean of which being 4.69 yrs ± 1.54 (Mean ± CI).



Distribution of Weight and Height in Donors

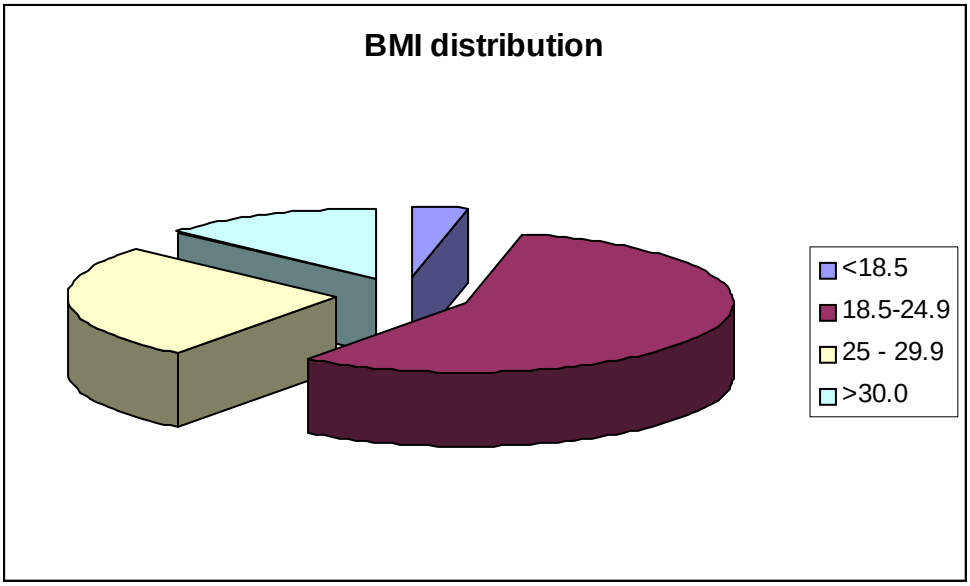
The average weight of the donors was 59.67 ± 3.75 kg (Mean ± CI).

The average height of the donors was 154.97 ± 2.36 cm (Mean ± CI).

The average BSA of the donors was 1.59 +/- 0.05 (Mean ± CI).

Distribution of BMI in Renal donors

BMI	Number
<18.5	1
18.5 - 24.9	17
25 - 29.9	8
>30.0	4



The average BMI of the donors was 24.84 ± 1.44 kg/m^2 (Mean ± CI).

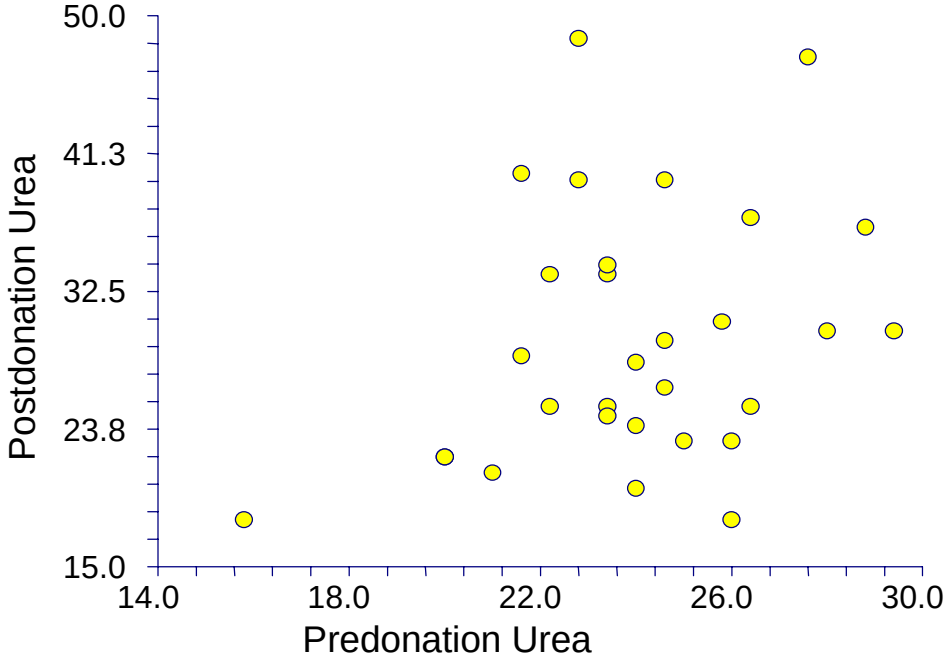
Distribution of Blood Urea in Donors Before and After donation

The average Predonation Urea was 23.96 mg% ± 1.04 and the mean Postdonation Urea was 29.48 mg% ± 2.91 (Mean ± CI)..

	N	Mean	95% of Lower Confidence Limit (LCL) of mean	95% of Upper Confidence Limit (UCL) of mean
Predonation Urea	10	23.96	22.893	25.026
Postdonation Urea	20	29.48	26.447	32.513

P=0.000543

A statistically significant elevation in blood urea levels was observed following donation of kidney ($p < 0.01$) on analysis with paired t test.

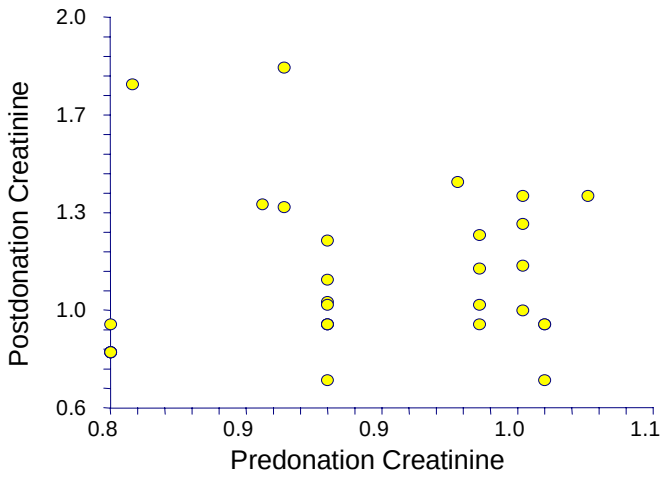


Distribution of Serum Creatinine in donors before and after donation

The mean Predonation Creatinine was 0.92 mg% ± 0.03 and the mean Postdonation Creatinine was 1.07 mg% ± 0.03 (Mean ± CI). An increase of 16% in serum creatinine was noted following renal donation over a mean follow up period of 4.69 years.

	N	Mean	95% of LCL of mean	95% of UCL of mean
Predonation Creatinine	30	0.915	0.888	0.942
Postdonation Creatinine	30	1.0693	0.9632	1.175

There was statistically significant elevation of serum creatinine in patient following the donation of kidney (p= 0.0065) as analyzed by paired t test.



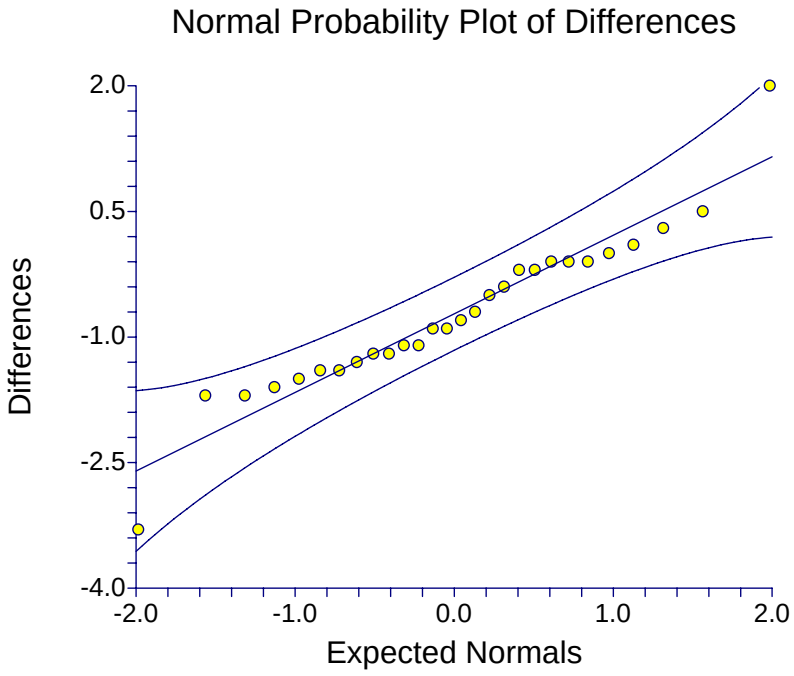
Distribution of Renal length and width assessed by USG

In my study, predonation data of 2 patients pertaining to kidney size was not available due to the unavailability of infrastructure at the time of transplantation. The mean predonation ultrasound length of the remnant kidney of the remaining 28 patients was 9.8 cm ± 0.3 (Mean ± CI).and the mean predonation ultrasound width of the remnant kidney was 3.82 cm ± 0.2 (Mean ± CI).

The mean postdonation ultrasound length of the kidney was 10.66 cm ± 0.26 and the mean postdonation ultrasound width of the kidney was 4.46 cm ± 0.21.

	N	Mean	95% LCL of Mean	95% UCL of Mean
Predonation Length	28	9.804	9.491	10.116
Postdonation Length	28	10.536	10.322	10.749

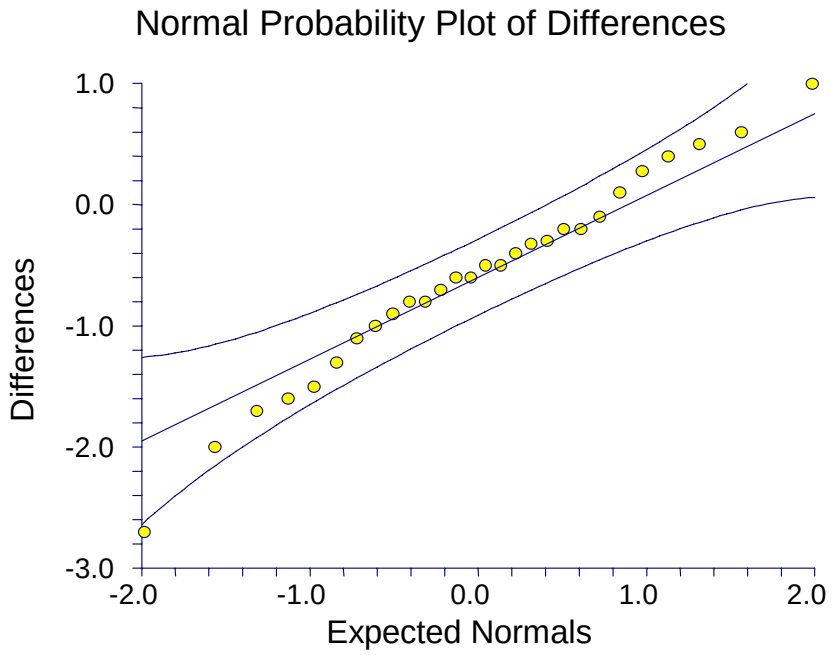
P=0.000413



	N	Mean	95% LCL of Mean	95% UCL of Mean
Predonation Width	28	3.821	3.616	4.027
Postdonation Width	28	4.426	4.206	4.646

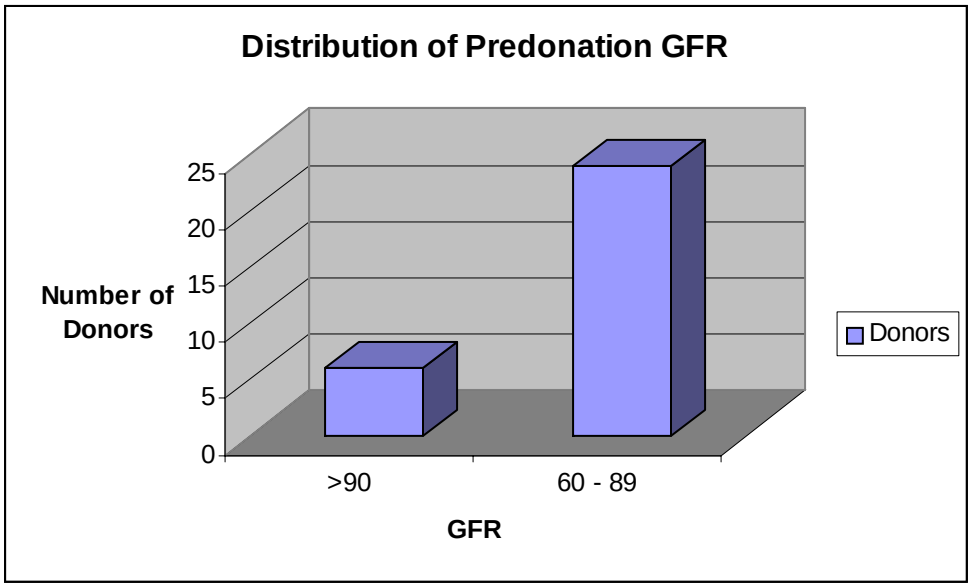
P=0.00067

A statistically significant increase in both renal length and width as evidenced by ultrasonography was observed following donation of kidney ($p<0.0001$).



Distribution of GFR prior to renal donation

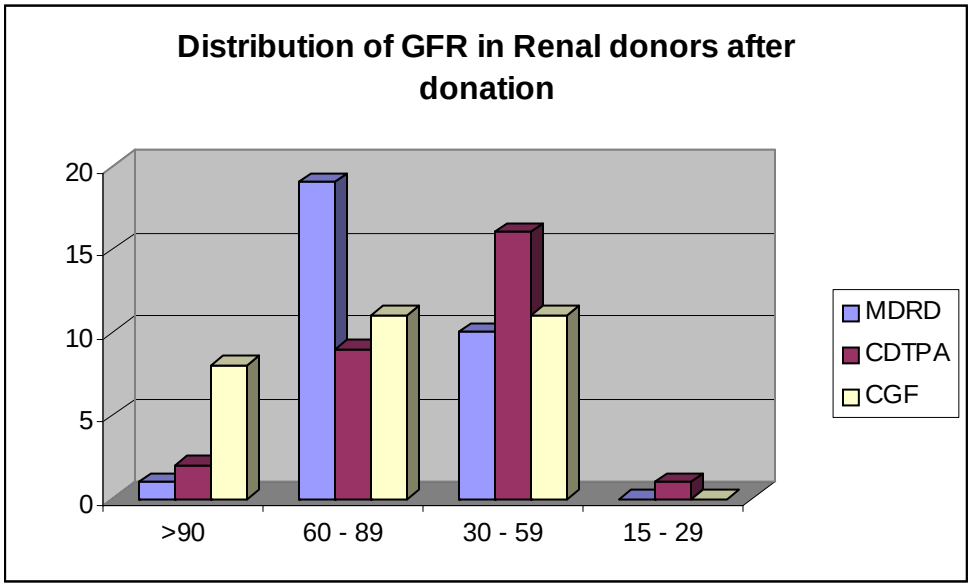
Predonation GFR using MDRD Formula	Number of Donors
>90	6
60 – 89	24



The mean Predonation GFR using the MDRD formula was 80.37 ml/min/1.73m^2 ± 5.9 (Mean ± CI).

Distribution of Postdonation GFR

CKD Stages	GFR Post renal donation		
	MDRD	CDTPA	CGF
>90	1	2	8
60 - 89	19	9	11
30 - 59	10	16	11
15 - 29	0	1	0



In this study, 2 of patients refrained from performing the DTPA test. The mean postdonation GFR estimated by Tc99 DTPA and corrected to 1.73 m^2 of body surface area of the remainder 28 patients was 57.78 ml/min/1.73m^2 ± 6.45 (Mean ± CI).

The mean postdonation kidney area as calculated by the Tc99 DTPA GFR estimation was 70.66 cm^2 ± 3.01 (Mean ± CI).

The mean perfusion index was 162.97 ± 30.7 (Mean ± CI).

The mean postdonation GFR calculated by Cockcroft Gault Formula was 72.18 ml/min/1.73 m^2 ± 7.79 (Mean ± CI).

The mean postdonation GFR calculated by MDRD Formula was 66.53 ml/min/1.73 m^2 ± 6.37 (Mean ± CI), which was observed to be 82.78% of the predonation GFR.

The mean reduction in GFR (calculated as per the MDRD formula) following renal donation was 14.17 ml/min/1.73m^2 ± 7.61 (Mean ± CI).

	N	Mean	95% LCL of Mean	95% UCL of Mean
Predonation GFR	30	80.367	74.637	86.096
Postdonation GFR	30	66.533	59.888	73.179

P=0.0012

The reduction in GFR following renal donation was found to be statistically significant on application of student's paired t test (p=0.0012).

Long-term outcomes of Renal donation

Reduction in GFR

Fall in GFR and Age at donation

	N	Mean	95% LCL of Mean	95% UCL of Mean
Age at the time of donation	30	37.067	33.731	40.402
Fall in GFR	30	-14.167	-22.114	-6.219

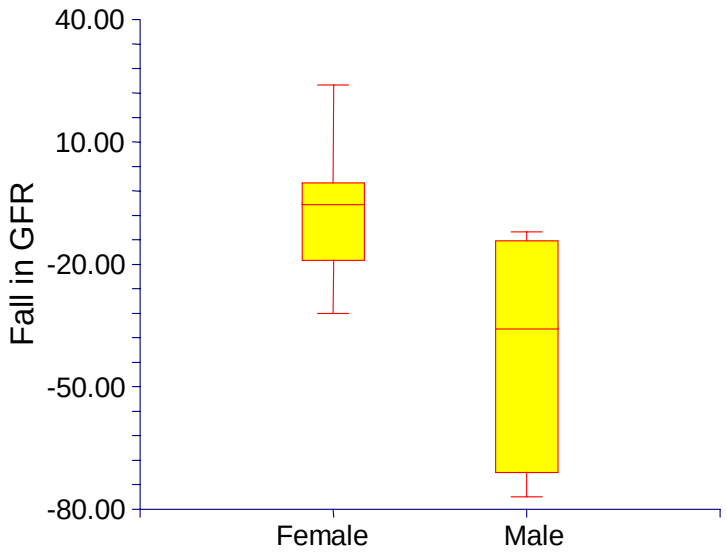
P<0.001

The fall in GFR was found to be statistically significant as the age at donation increased. ($p<0.001$)

Fall in GFR and Gender

Gender	N	Mean	95% LCL of Mean	95% UCL of Mean
Female	24	-7.542	-13.256	-1.827
Male	6	-40.667	-69.049	-12.284

A statistically significant fall in GFR was noted among males in comparison to females (p<0.001) when analysed using student's t test.



Fall in GFR and BMI

	N	Mean	95% LCL of Mean	95% UCL of Mean
BMI	30	24.843	23.341	26.346
Fall in GFR	30	-14.167	-22.114	-6.219

P<0.001

An increase in BMI was associated with a statistically significant reduction in GFR (p<0.001).

Fall in GFR and BSA

	N	Mean	95% LCL of Mean	95% UCL of Mean
BMI	30	24.843	23.341	26.346
Fall in GFR	30	-14.167	-22.114	-6.219

A statistically significant correlation was noted between reduction in GFR and body mass index ($p<0.0001$).

Fall in GFR and Predonation GFR

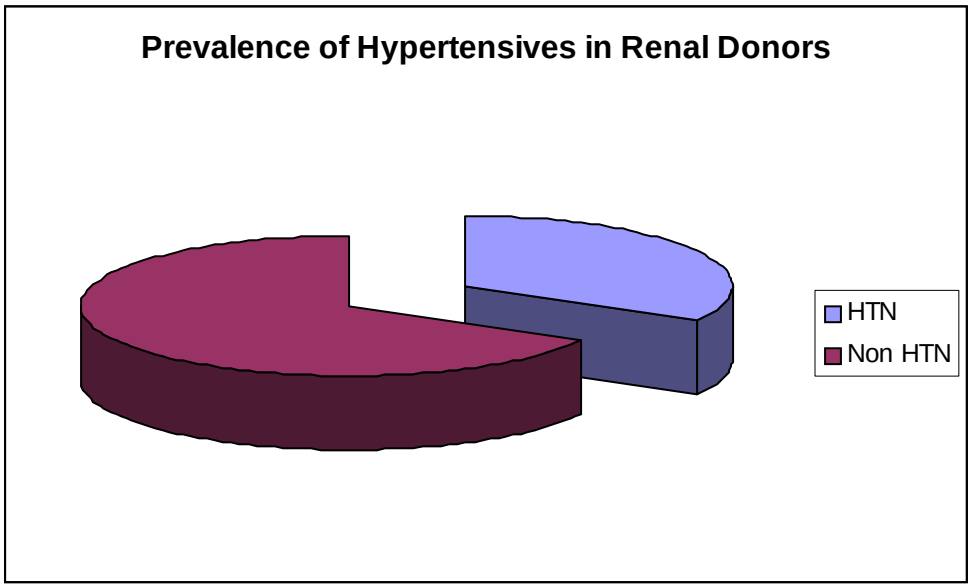
	N	Mean	95% LCL of Mean	95% UCL of Mean
Predonation GFR	30	80.367	74.637	86.096
Fall in GFR	30	-14.167	-22.114	-6.219

P<0.0001

There is a statistically significant correlation between predonation GFR and reduction in GFR ($p<0.0001$) on application of students t test.

Hypertension

The number of donors on antihypertensive drugs was 4 and the number of newly detected hypertensives was 6. Therefore, 10 patients out of the sample population was hypertensive following renal donation.



There was a 33% increased incidence of hypertension in patients following renal donation, which was found to be statistical significant ($p < 0.00001$).

Age group	No. of Hypertensives	No. of Non-hypertensives	% of hypertensives
35 – 49	7	15	31.8
50 – 64	3	1	75

		Hypertensive	Non Hypertensive	Total
Chennai Population	35 – 49 yrs	21%	79%	100%
	50 – 64 yrs	38.4%	86.6%	
Donated Population	35 – 49 yrs	31.8%	68.2%	100%
	50 – 64 yrs	75%	25%	

When the above-mentioned population was age-matched with data from Dr V Mohan's CURES study (n = 2350), which investigated the prevalence of hypertension in Urban Chennai population statistical significant prevalence of hypertension was demonstrated only in the age group between 50 – 64 yrs ($p < 0.0001$, RR = 1.953). In the age group between 35 – 49 yrs, although a 1.54 times increased risk of development of hypertension was demonstrated in nephrectomised patients compared to the normal population, no statistical significance was noted ($p = 0.1160$).

Hypertension and Gender

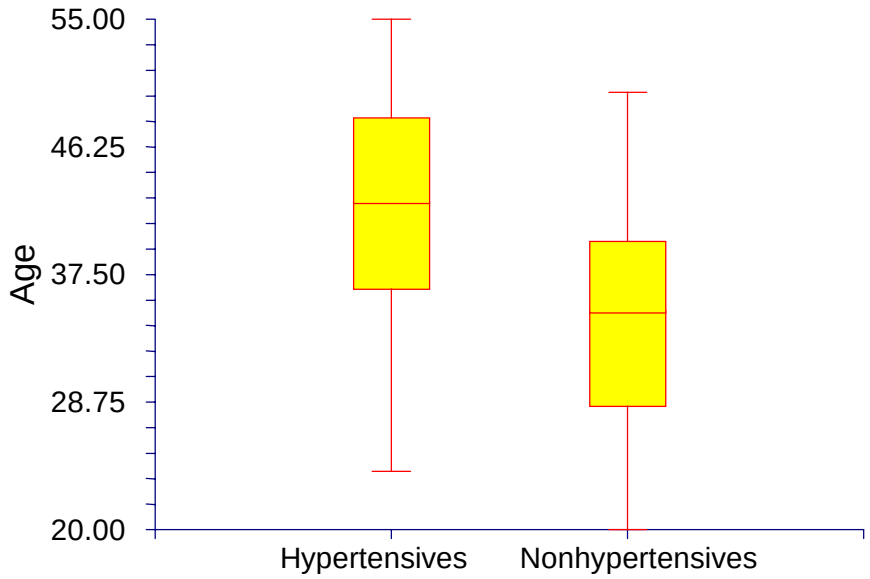
Gender was not found to be a factor affecting development of hypertension in renal donation patients. The Fischer exact test was statistically insignificant ($p= 0.1413$)

Sex	Hypertensive	Non Hypertensive	Total
Female	6	18	24
Male	4	2	6
Total	10	20	30

Age and Hypertension

The mean age of patients who were hypertensive was 42.2yrs and a statistically significant correlation between age at donation and development of hypertension was seen in donors (p=0.023197).

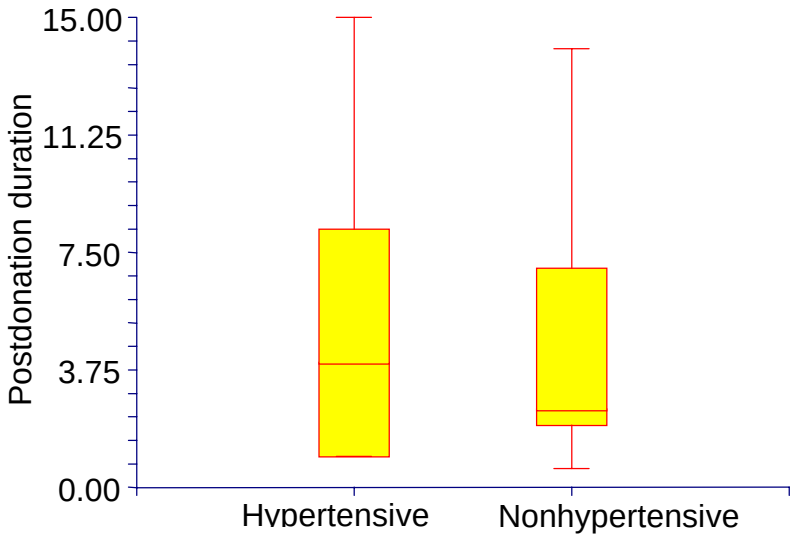
	N	Mean	95% C.I	
			LCL	UCL
Hypertensive	10	42.2	35.6215	48.79785
Nonhypertensive	20	34.5	30.85416	38.14384



Hypertension and Postdonation period

	N	Mean	Std. Deviation	Std. Error	95% of LCL of Mean	95% of UCL of Mean
Hypertensive	10	5	4.643	1.468	1.679	8.321
Nonhypertensive	20	4.54	4.217	0.943	2.566	6.513

No statistically significant correlation was found between the development of hypertension and the postdonation duration (p=0.787222).



Body Mass Index and Hypertension

	N	Mean	95% of LCL of mean	95% of UCL of mean
Hypertensive	10	26.3	22.429	30.171
Nonhypertensive	20	24.12	22.698	25.532

P= 0.165

There was no statistically significant correlation observed between the development of hypertension and the BMI of the patients (p=0.165) on application of student's t test.

Body Surface Area and hypertension

	N	Mean	95% of LCL of mean	95% of UCL of mean
Hypertensive	10	1.659	1.562	1.755
Nonhypertensive	20	1.55	1.490	1.609

P= 0.038

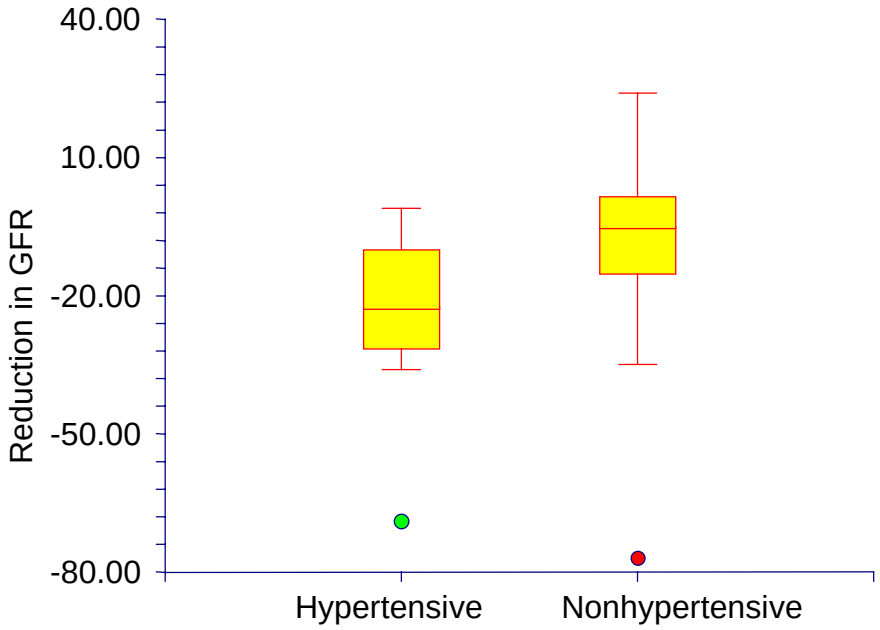
Statistically significant correlation was observed between Body surface area and the development of hypertension (p=0.038) on application of student's t test.

Reduction in GFR and hypertension

	N	Mean	95% LCL of Mean	95% UCL of Mean
Hypertensive	10	-23.8	-37.662	-9.9377
Nonhypertensive	20	-9.35	-19.170	0.470

P=0.0792

Although hypertensive patients had a greater reduction in GFR compared to their nonhypertensive controls, this difference was not found to be statistically significant (p=0.0792).



Hypertension and Postdonation GFR

	N	Mean	95% LCL of Mean	95% UCL of Mean
Hypertensive	10	56.5	42.344	70.656
Nonhypertensive	20	71.55	64.632	78.468

P=0.0262

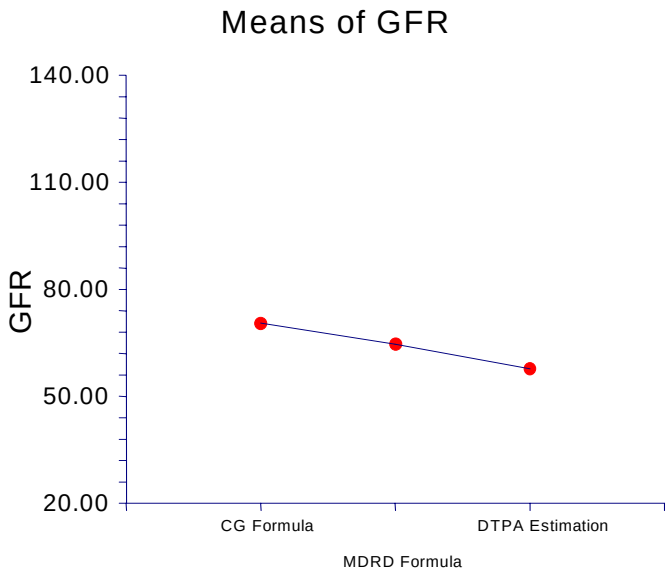
A statistically significant correlation was observed between hypertension and postdonation GFR(p=0.0262).

Comparison of GFR by using different formula in patients with single kidney

GFR	N	Mean	Standard Error
GFR by Cockcroft Gault Formula	28	70.493	3.53
GFR by MDRD Formula	28	64.643	3.53
GFR estimated by 99mTc DTPA	28	57.779	3.53

P= 0.043882

A statistically significant difference was established between 3 methods of calculation of GFR on analysis with Repeated measures Analysis of Variance (p= 0.0439).



KEY

- S. No. – Serial Number
- AHTN Drugs – Antihypertensive drug therapy
- Wt. – Weight (in kilograms)
- Ht. – Height (in centimeters)
- BMI – Body Mass index (in kg/m^2)
- BSA – Body Surface Area (in m^2)
- SBP – Systolic Blood Pressure (in mm of Hg)
- DBP – Diastolic Blood Pressure (in mm of Hg)
- B. Urea – Blood Urea (in mg%)
- S. Cr. – Serum Creatinine (in mg%)
- USG L – Ultrasound length of kidney (in centimeters)
- USG W – Ultrasound width of kidney (in centimeters)
- GFR MDRD – Glomerular Filtration Rate estimated by MDRD (IV) formulae (in ml /min /$1.73m^2$)
- UDTPA – Glomerular Filtration Rate estimated by 99Tc DTPA not corrected to $1.73m^2$
- CGF – Glomerular Filtration Rate estimated by Cockcroft Gault Formula (in ml /min / $1.73m^2$)

- CDTPA – Glomerular Filtration Rate corrected to 1.73m2 body surface area (in ml/ min /1.73m^2)
- ⇓ GFR– Reduction in Glomerular Filtration Rate

Predonation					Post donation								⇓ GFR	Renal Area	S. No.
B.Urea (mg%)	S. Cr. (mg%)	USG L (cm)	USG W (cm)	GFR MDRD	B. Urea (mg%)	S. Cr. (mg%)	USG L (cm)	USG W (cm)	GFR						
									UDTPA	CGF	MDRD	CDTPA			
20	0.8	9.5	3.6	87	22	0.8	11.1	5.6	44.09	91.2	86	174.31	-1	47.97	1
25	1	10.4	3.9	69	23	0.9	10.6	4.5	61.04	102.4	78	240.75	9	62.12	2
15.8	0.8	NA	NA	94	18	0.8	12.4	4.5	61.75	102.8	86	72.24	-8	63.22	3
20	0.8	9.9	4.8	69	22	0.9	9.8	4.7	45.22	57	68	175.82	-1	53.22	4
26	1	10.5	3.8	71	18	0.7	10.6	5.1	50.64	135.9	67	154.85	-4	49.22	5
22.8	0.88	9.6	4.3	100	48.6	1.82	10.8	4.8	27.02	59.1	31	417.99	-69	24.22	6
21.6	0.88	10	4.4	76	40	1.32	10.5	5.2	45.69	56.8	46	182.05	-30	48.49	7
28	1	9.9	3.5	64	30	0.9	10.6	4.2	53.71	72.4	71	139.96	7	58.1	8
22.8	0.9	7.2	2.5	76	39.6	1.2	10.5	5.2	39.03	58.3	54	65.96	-22	44.13	9
23.4	0.9	9.7	3.6	73	25.2	0.9	10.6	4.5	30.8	82.7	73	206.5	0	32.89	10
24	0.9	8.9	4	77	20	0.7	10	4.6	DNP	99.2	101	DNP	24	DNP	11
26	0.8	10.3	4.1	87	23	0.8	12	4.2	DNP	92.4	85	DNP	-2	DNP	12
26.4	0.96	10.6	3.9	67	37.2	1.41	10.1	4.1	38.68	62.7	41	315.22	-26	37.81	13
24.6	0.99	10.1	3.1	65	39.6	1.36	11.5	4.1	48.81	55.6	45	125.39	-20	49.67	14
25.8	0.9	9.1	3	114	30.6	1.06	10.6	4.7	61.89	106.4	89	115.93	-35	66.92	15
22.2	0.99	9.8	3.9	63	25.2	0.95	10.2	4.4	55.84	60.5	65	285.82	2	62.5	16
23.4	0.99	11.3	4.5	83	33.6	1.26	9.3	4	52.1	60.89	47	253.52	-36	60.89	17
26.4	0.9	9.3	3.8	101	25.2	0.98	10.4	4.9	53.28	90.7	89	100.28	-12	53.2	18
23.4	0.97	9.4	3.2	69	24.6	0.9	11.1	3.6	53.6	64.2	74	157.96	5	67.19	19
22.2	0.97	9.1	4.2	68	33.6	1.22	10	3.2	72.03	40.6	52	93.29	-16	93.69	20
24.6	0.99	10.8	3.6	102	29.4	1.11	11	3.9	71.3	78.4	87	69.78	-15	73.42	21
28.8	1.02	10.2	3.6	71	36.6	1.36	11.4	5.2	49.15	59.6	46	81.29	-25	51.22	22
27.6	0.81	9.2	3.6	124	47.4	1.76	10.5	5.1	53.64	46.4	47	152.24	-77	58	23
23.4	0.87	8.8	3.2	77	34.2	1.33	10.2	4	42.96	44.9	45	189.8	-32	49.22	24
24	0.9	10.1	4.1	75	28	0.9	10.2	4.3	54.71	59.4	75	164.72	0	70.63	25
21	0.8	NA	NA	89	21	0.8	12.5	5.5	85.8	58.9	82	198.78	-7	114.18	26
24	0.9	9.5	4.2	72	24	0.97	10.3	3.92	42.86	65.2	66	73.13	-6	46.34	27
21.6	0.97	10.1	4.7	72	28.4	1.1	10.2	4.1	48.17	62.6	61	114.59	-11	53.76	28
24.6	0.97	10.9	3.6	69	26.4	0.97	10.6	3.92	60.87	63.4	65	80.22	-4	67.94	29
29.4	0.9	10.3	4.3	87	30	0.9	10.3	3.9	52.65	74.8	74	160.65	-13	57.65	30

MASTER CHART

S.No	Age	Age at donation	Sex	Post. Duration	Recipient	AHTN Drugs	Wt. (kg)	Ht. (cm)	BMI (kg/m^2)	BSA (m^2)	SBP (mm Hg)	DBP (mm Hg)
1	37	35	F	2	Husband	-	60	156	24.7	1.59	110	80
2	30	28	F	2	Husband	-	71	155	29.6	1.7	120	70
3	36	23	F	13	Elder Brother	-	67	159	26.5	1.69	110	70
4	58	55	F	3	Son	+	53	150	23.6	1.47	140	80
5	34	27	F	7	Husband	-	76	158	30.4	1.78	110	80
6	52	43	M	9	Elder Brother Daughter	+	88	163	33.1	1.93	130	80
7	48	40	F	8	Sister	+	69	149	31.1	1.63	110	80
8	48	43	F	5	Daughter	-	60	157	24.3	1.6	120	70
9	36	34	F	2	Daughter	-	57	152	24.7	1.53	90	60
10	43	41	F	2	Daughter	-	65	153	27.8	1.62	112	80
11	35	32	F	3	Elder Brother	-	56	150	24.9	1.53	110	70
12	39	35	F	4	Mother Sister	-	62	154	26.1	1.63	122	86
13	46	45	F	1	Son	+	78	155	32.5	1.77	120	80
14	43	42	F	1	Son	-	66	162	25.1	1.7	140	90
15	27	20	M	7	Father	-	58	158	23.2	1.6	110	80
16	53	50	F	3	Son	-	56	155	23.3	1.54	122	80
17	56	55	M	1	Daughter	-	49	161	18.9	1.5	110	100
18	43	37	M	6	Brother	-	66	158	26.4	1.7	150	80
19	38	37	F	1	Son	-	48	146	22.5	1.38	130	60
20	40	38	F	2	Son	-	42	150	18.7	1.33	98	80
21	40	35	M	5	Mother	-	54	178	17	1.68	122	90
22	39	24	F	15	Brother	-	68	154	28.7	1.66	140	80
23	35	24	M	11	Brother	-	56	163	21.1	1.6	120	80
24	48	39	F	9	Brother	-	55	153	23.5	1.51	118	70
25	37	37	F	0.6	Son	-	44	147	20.4	1.34	110	80
26	45	31	F	14	Unknown	-	42	146	19.7	1.3	110	80
27	46	46	F	0.6	Son	-	57	156	23.4	1.6	110	82
28	32	30	F	2	Brother	-	54	155	22.5	1.55	130	80
29	47	46	F	1	Son	+	56	145	26.6	1.55	118	80
30	40	40	F	0.6	Son	-	57	151	25	1.58	124	80